operative adhesions. The abdominal wall and localized abscesses of the peritoneal cavity should be drained, as he suggests, with small Penrose strips.

The essayist has done well to remind us that the best surgery is the least that can be done to eliminate continued reinfection of the peritoneal cavity. I agree with him that spinal anesthesia is a real boon to the surgeon, but in these days when its popularity is increasing so tremendously, it is well for its friends to warn against its use in those patients so ill that any anesthetic or surgical procedure is likely to lead to a fatal outcome. Spinal anesthesia cannot be asked to perform miracles of healing, and only disrepute can come from its use upon moribund or extremely weak patients with low blood pressure.

In the postoperative treatment of patients with acute abdominal conditions, it is gratifying to note the unanimity with which surgeons of experience lay emphasis upon the two all-important, life-saving elements, namely, a high normal saline solution intake and continuous gastric lavage. A parenteral administration of 4000 cubic centimeters a day should be insisted upon. The fluid should all have a normal salt content of chlorid, and whether dextrose should be added depends upon the duration of treatment and the patient's consequent need of carbohydrate. One thing to remember is that a patient's circulation can never be overloaded with solution given subcutaneously. The chlorid loss through vomiting or continuous gastric or duodenal lavage is always high, but the stomach and upper intestines must be kept empty if peristalsis is to be reëstablished. This can best be done, as Doctor Butler states, not by repeated and exhausting stomach washes with a large tube, but by an indwelling, transnasal Levin duodenal tube, to which continuous mild suction is applied. By use of this method it is no longer necessary to guess when peristalsis is reëstablished or when food administration can safely be started.

OPTIC NERVE CHANGES IN NONTRAUMATIC NEUROLOGIC DISORDERS*

REPORT OF CASES

By WALTER F. SCHALLER, M. D.
San Francisco

DISCUSSION *by Otto Barkan, M. D., San Francisco, Joseph L. McCool, M. D., San Francisco.*

SCARCELY any neurological examination, apart from the peripheral nervous system, is complete without some idea of the state of the optic nerve. A proper neurological status includes examination of the vision, visual fields, appearance of the nerve head, and pupillary reactions. This has been a routine in private neuropsychiatric case records now numbering 4660. It has been of considerable personal interest and profit to review these case records, particularly in the light of incidence, diagnosis, and course. A paper of this character cannot of necessity be comprehensive, and I fear will be discursive, but it will at least represent the clinical experience of a neurologist over a twenty-year period. Papilledema, the Argyll Robertson phenomenon, optic neuritis and optic atrophy, and certain visual syndromes are considered.

PAPILLEDEMA

Papilledema, or choked disk, is characterized at the onset by vascular changes: contraction of arteries, tortuosity and dilatation of veins, and projection of the papilla; this progresses to a state of edema of the disk and surrounding retina with hemorrhages and white streaks of irregular patchy degeneration in and about the disk. Important is the long retention of acuity of vision with periods of dimness of vision and transitory blindness, first emphasized by Jackson.

Since Albrecht v. Graefe (1860) published his work on papillary stasis over seventy years ago, this condition has been the subject of much research and discussion, resulting in four principal theories, namely, of venous stasis; transport theory of the optic sheath; extension of brain edema; and the optic nerve lymph stasis theory. Wildbrand and Saenger [1] (1909) in their system devote two hundred eighty-one pages to papillary stasis, thirty-one of which are devoted entirely to theoretic discussion. The discussion still continues, but the tendency in later years has appeared to favor either the optic sheath theory or the lymph stasis theory (C. Behr), the latter explaining cases with and without intracranial pressure. Claude, Lamarche, and Durbar [2] state that intracranial pressure is accompanied by arterial hypertension of the retinal vessels, which condition is established by precise determination. When the equilibrium thus established is disturbed by a lowering of the retinal arterial pressure, signs of papillary stasis occur. In three cases the authors have found this fall in retinal pressure to precede the ophthalmoscopic evidence of stasis, calling attention to this fact without offering a definite theory of its mechanism. Nevertheless they favor a reflex vasomotor explanation to the mechanical theory.

L. Dupuy-Dutemps [3] has been concerned with the subject of pathogenesis since his Paris thesis of 1900. He believes papilledema is due to a compression of the central vein of the optic nerve with consequent venous stasis. In conditions of intracranial pressure the compression is effected by transmitted pressure to the optic sheath, affecting the vein at the exit from the optic nerve, about one centimeter from the retina; but local conditions may affect the vein retro-orbitally in the optic nerve itself, and also produce papillary stasis. Tilney [4] describes a canal from the third ventricle and lined by ependyma which extends laterally over the optic chiasm and optic nerves. He names this the supra-optic canal. Distention of this canal, by lesions obstructing drainage, is advanced as a cause of papilledema. It is the condition of intracranial pressure which particularly concerns the neurologist, and for the presence or absence of which he seeks information from the eyeground examination; however, an appreciable intracranial pressure may exist, even reported as high as 400 to 500 millimeters of water, without accompanying papillary stasis.

REPORT OF A CASE

The following case of spontaneous subarachnoid bleeding is significant in indicating the degree of intracranial pressure at which one may expect eyeground changes.

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* Read before the Eye, Ear, Nose, and Throat Section of the California Medical Association at the sixtieth annual session at San Francisco, April 27-30, 1931.

A male in the late forties was seized with a sudden onset of collapse and a complete persisting third nerve paralysis. A month later, after a night on a train, he again showed signs of collapse, complaining of severe headache, the pulse rate dropping to forty. An examination of the eyes showed a distinct edema of the left disk with one large and two lesser hemorrhages. Lumbar puncture revealed a zanthrochromic fluid under a pressure of 390 millimeters of water, without other important serological findings. The patient improved, the papilledema receded and has not recurred. A recent fluid pressure estimation was normal.

It appears to be a well-established fact that during increase of intracranial pressure with papilledema the disk swelling may recede in one or both eyes to a condition of atrophy without disk elevation. Farisotti [5] has explained this recession and atrophy by obliteration of the intravaginal space and replacement by fibrous tissue.

In recent years much interest has been directed to and a considerable literature has accumulated on the relationship of paranasal sinus disease to optic nerve stasis. When optic nerve stasis occurs, a brain tumor may be overlooked and a misdirected operation performed on the sinuses. An observation in point is here reported:

REPORT OF A CASE

A truck driver, age thirty-four, with a history of frontal and temporal headache and dizziness of several years' duration, consulted an oculist for dimness of vision seven months before he came under my observation. Papilledema of both disks, somewhat less than three diopters, and contraction of the visual fields were found. Pus was found in the left ethmoid cells and sphenoid cells, which were opened and drained, without, however, relieving the papilledema. Six months after this operation, when I first saw the patient, the condition had progressed to complete loss of vision. Bilateral papilledema, pathological pyramidal tract reflexes in the lower extremities, a higher pitched note on the left side of the skull, and the recent occurrence of Jacksonian epilepsy, commencing in the right upper extremity, definitely indicated cerebral pathology. The cerebrospinal fluid was under 850 millimeters of pressure. X-ray studies revealed some destruction of the dorsum of the sella and a very marked erosion of the floor. The symptoms were indicative of a left frontal brain tumor, and operation being decided upon, a left osteoplastic flap was made over this region. Brain puncture opened into a gliomatous cyst, from which about sixty-five cubic centimeters of yellowish fluid was removed. The patient made a good operative recovery, although he suffered a partial postoperative motor aphasia. The complicating sinus infection obscured the condition of brain tumor, which might have been discovered months earlier by skull studies and an estimation of the cerebrospinal fluid pressure, and blindness prevented by relieving intracranial pressure.

The ophthalmologist and the otologist must, moreover, ever keep in mind that a papillary stasis of central origin may be unilateral, and may be unaccompanied at the onset by intracranial focal symptoms and signs.

I have maintained for years that a diagnostic lumbar puncture, undertaken with proper precautions in suspected brain tumor cases, is of such aid in diagnosis as to outweigh the danger of herniation of the medulla into the foramen magnum. I have never had such an experience, and have not hesitated to perform a puncture, provided that the diagnosis was uncertain and the test would give important information. The patient should be in a reclining position and only a few cubic centimeters of fluid slowly withdrawn, noting any sudden fall of pressure by means of a manometer. Afterward, the foot of the bed should be elevated. So that no fluid will be unnecessarily withdrawn or lost, I use a Claude's pressure gauge. In a recent case of typical acusticus tumor, successfully operated by Cushing, with bulbar symptoms and signs of intracranial pressure, such as somnolency, mental lassitude, headache, especially on coughing and straining, but with no indications of papillary stasis, I withheld from puncture, but did so because the diagnosis was undoubted, not because I should have been hesitant to attempt it if the diagnosis had been in doubt.

Disk changes in acute epidemic encephalitis may occur as hyperemia, haziness, or a mild neuritis, rarely a true papilledema. That this, however, may occur has been reported by good authority in verified cases, and in a series of 235 encephalitic cases I observed two such examples. A similar situation exists in multiple sclerosis, which may be described, pathologically speaking, as a disseminated encephalitis. In my series of twenty cases of multiple sclerosis I observed no case of papilledema. Papilledema occurs most frequently in brain tumor, obviously because of the frequent occurrence of increased intracranial pressure. Two instances only of papilledema occurred in six verified brain abscesses. In a moribund patient with cerebral symptomatology of a week's duration an occipital abscess had ruptured into the ventricle without evidence of papilledema. In another case of verified cortical abscess with fatal outcome twelve days from onset, with definite involvement of the subarachnoid space, there was no evidence of disk changes. In both these cases the abscesses evidently decompressed into the ventricular and subarachnoid spaces, thus preventing internal hydrocephalus.

A papilledema of alarming degree may result from a cranial operation, as was the case following left mastoidectomy in a child. Sinus injury was suspected because of after-bleeding. A sinus clot one inch long was removed in a second operation, and the jugular vein ligated. Material from the vein was reported as a diplostreptococcus thrombosis. Six weeks following operation a paresis of the left external rectus and papilledema on the right side were noted, and a few days later papilledema appeared on the left side. The swelling increased to four diopters in both eyes. There were no other alarming physical signs; the disks finally receded, and in two months they were flat and the eyes regained normal function. Circulatory disturbance in the posterior fossa and consequent edema best explained the phenomena.

A brain tumor may cause papilledema without apparently being large enough to add materially to the contents of the cranial cavity or to inter-

fere with the flow of cerebrospinal fluid. Such a condition occurred in a patient presenting a definite astereognosis in one upper extremity, enabling the localization and removal of a hazelnut sized tumor from the parietal cortex. The reason for such an occurrence is obscure and has been discussed by Spiller; it is a fact which has to be considered in the pathogenesis of papilledema.

ARGYLL ROBERTSON PUPIL

It is generally conceded that the optic nerve contains fibers concerned with light perception, as well as fibers concerned with visual images. The light fibers at first run a course similar to the visual fibers, decussating in the chiasm, but later separate from the visual fibers in the diencephalon, branching off before the external geniculate body is reached, to pass into the superior brachium (Karpus and Kreidl) to form as the afferent sensory arm a reflex arc with the sympathetic third nerve nucleii (Edinger-Westphal nucleii) by way of the superior colliculi and colliculonuclear tract. The crossed fibers of the latter (fountain decussation of Meynert) skirt the aqueduct of Sylvius. The hypothesis has been offered that these afferents are affected by a toxic spread, as in syphilis, through subependymal tissues, producing the Argyll Robertson pupil. The Argyll Robertson pupil has been reported in mesencephalic tumors, multiple sclerosis, alcoholism, epidemic encephalitis, and in both ocular and cerebral traumatism, so therefore is not to be regarded as unquestionably of syphilitic nature or as having a precise and constant pathological localization. The motor efferents from the third nerve sympathetic nucleii pass to the iris by way of the ciliary ganglion. The course of the fibers is ill defined, and Wilson [6] is authority for the statement that "Lesions of the oculomotor trunk nerve never cause reflex iridoplegia." Sven Ingvan [7] cites experimental and pathological evidence that light fibers run in the outer layers of the optic nerves and tracts and on the surface of the diencephalon. He quotes various authors to the effect that optic atrophy begins in the marginal region of the optic system. He found such to be the case in the pathological study of an early case of tabes with Argyll Robertson pupils and believes that degeneration of the superficially placed light fibers occasions the phenomenon.

Bing [8] places the lesion of the Argyll Robertson pupil between the primary visual neurone and the sphincter nucleii of the third nerve, for, he states, if the lesion were proximal to this area, a hemianopsia would occur; if distal, sphincter paralysis.

Spiller [9] reports a case obviously of tabes in which there was a left unilateral Argyll Robertson pupil, the right pupil contracting promptly to direct and consensual light stimulus. The affected eye, other than the light reflex iridoplegia, was normal except for variation in the size of the pupil and a paradoxical pupillary reflex. The certainty that light impulses from the left eye reached the right oculomotor nucleus was clinical evidence to Spiller that there must be a center in the oculomotor nucleus on each side concerned with pupillary action on the same side (Edinger-Westphal nucleus).

The description of Douglas Argyll Robertson in 1869 of the dissociated pupillary reaction which bears his name has scarcely been improved upon. His astute clinical observations include myosis, which he regarded as essential to his sign, and the preservation of good vision, noting the fact that there was ability to read fine print. The implication, therefore, was that the accommodative power or ciliary muscle function was intact. In one hundred and five personal cases of tabes the light reaction was impaired in seventy cases, myosis was noted in eleven, and mydriasis in seven. In seven cases with myosis the light reaction was impaired; in one the pupils reacted to light, three not noted. In the cases with mydriasis there were three in which the light reaction was normal and four cases in which it was impaired. In one case one eye was definitely myotic, the other definitely mydriatic, tabes alone apparently accounting for this condition. It is justifiable to diagnose an Argyll Robertson pupil as one in which the light reflex is definitely impaired, though not abolished, and in which the accommodation-convergence reaction is normal. Otherwise one must assume a sudden and complete light iridoplegia, not a reasonable assumption in a chronic condition, such as neurospyhilis.

Mehrtens and Otto Barkan [10] have studied the pupillary reaction in epidemic encephalitis by means of the Hess pupilloscope. In a series of thirty-six cases they found no example of the Argyll Robertson pupil and a doubtful iridoplegia in but one case. They therefore question the occurrence of the Argyll Robertson sign in epidemic encephalitis. Isolated reduction of the accommodative power (ciliary muscle function) was found in thirteen cases in their series, being present also in sixteen cases of ophthalmoplegia interna; in all, the accommodative power was affected in 80 per cent of the cases and was, therefore, considered the characteristic ocular sign in epidemic encephalitis. My material of two hundred and thirty-five cases is in general confirmatory to this view—in thirty-two cases blurred vision was a symptom clinically interpretative of impaired ciliary function. Four cases showed iridoplegia and five cases disturbed accommodation-convergence reaction (in one case lost) with normal light reflex—reversal of the Argyll Robertson phenomenon. There was but one case of Argyll Robertson pupil, and this was unilateral. In all, thirty-three patients showed disturbed light reactions. Associated with pupillary abnormalities, miosis occurred in six cases, mydriasis in fourteen. Dissociated iridoplegia, therefore, was rare, and the Argyll Robertson sign the least frequent of eye findings, occurring in less than one-half per cent of all cases. The various states of the pupil in encephalitis can be

reasonably explained by the disseminated nature and inconstant localization of the encephalitic lesion, thus differing from a systemic and meningeal pathology found in tabes.

In thirty-six cases of chronic alcoholism fourteen cases showed disturbed pupillary light reflex, but in only two was this reflex entirely abolished. The pupillary disturbance is generally not an isolated phenomenon, but associated with other signs of peripheral neuritis, such as lost or diminished ankle jerks and diminished tactile sensation. Dependent upon alcoholic withdrawal, the prognosis is generally excellent, as the following case report will testify:

REPORT OF A CASE

A male in the middle forties when observed in April presented a mental condition characteristic of chronic alcoholism and physical complaints of occasional gastric crises and neuritic pains. His pupils were sluggish to light, especially one pupil; the right ankle jerk was lost, the left diminished. Previous to this examination the analysis of the cerebrospinal fluid was negative. His blood Wassermann was negative. After a six months' abstinence from alcohol his pupils reacted promptly and equally to light and his ankle jerks responded normally.

OPTIC NEURITIS AND OPTIC ATROPHY

A general discussion of these subjects is of too large scope to be fully attempted in this paper. Among the recent noteworthy contributions to optic neuritis is the scholarly paper of Walker [11] read last year before this section.

Thirty-four cases of optic neuritis are recorded in my series; seventeen of these occurred in a total of twenty cases of multiple sclerosis in which either atrophy, central scotoma, or transient blindness was noted. Four cases occurred in encephalitis. In eleven cases no definite cause of the condition was found, representing, therefore, a discouragingly high incidence of unknown etiology.

REPORT OF A CASE

A case illustrating methyl alcohol poisoning and amblyopia occurred in a temperate man, age sixty-one, who obtained his alcohol from a "high-grade bootlegger." Several persons who were likewise provided and partook of this same supply were unaffected, so he did not attribute his loss of sight, which came on within one day, to this case. Chemical analysis of a sample of the alcohol, however, showed it to contain in the neighborhood of 50 per cent methyl alcohol. Two days before the onset of visual disturbance he felt a malaise, suffered from slight headache, and went to sleep going home on a ferry-boat. However, he had no other important neurological symptoms. When he was seen six days after onset of visual disturbance there was some light perception and some slight disk edema. Light perception increased, but useful vision was lost. In this case there appears to have been a selective action of a moderate amount of methyl alcohol in a susceptible individual.

REPORT OF A CASE

A case illustrating an optic neuritis due to paranasal sinus disease is the following:

A woman, age thirty-nine, complained of rapid loss of sight in the right eye following a severe head cold a month previously. An oculist found complete blindness in this eye and slight pallor of the disk. The pupil was slightly dilated. Other neurological complaints were stiffness and cramp in the legs, numbness in the right hand, and trigeminal numbness. After images and visual hallucinosis had occurred in the period of failing vision. The general neurological examination was essentially negative. Local investigation of the sinuses by an otologist revealed no definite evidence of disease. Special x-ray films of the right optic foramen showed the roof to be so indistinct as to be hardly discernible contrasted with the normal appearance of the left optic foramen. On this x-ray evidence the sphenoids were opened, revealing thickened membranes and pus. Eight days following the operation fingers could be counted, and five weeks later large objects recognized. The optic disk at this time showed a total pallor.

OPTIC ATROPHY

In the tabetic series (one hundred five cases) optic atrophy was noted in twelve cases; in the encephalitis series (two hundred thirty-five cases) no definite case of atrophy is recorded, but a neuritis was observed in four cases. Sands[12] reports a case of optic atrophy due to encephalitis and quotes other observed cases. In three of my cases the cause of atrophy was undetermined. In the multiple sclerosis series (twenty cases) disk pallor was noted in thirteen. There was one case of traumatic atrophy. The complication of optic atrophy in neurosyphilis brings up the question of the danger of arsenical and particularly tryparsamid therapy.

In rare cases tryparsamid has been incriminated in the production of optic atrophy. Woods and Moore,[13] studying visual disturbances in tryparsamid therapy, state that "Preëxisting syphilitic disease of the optic nerve is not necessarily a contraindication to the use of tryparsamid." In their series of one hundred ten patients whose eyes were examined before and during treatment, twelve had preëxisting disease of the optic nerve, and in these visual disturbances occurred in four. Cody and Alvis[14] believe that patients with optic involvement "are more liable to injury than normal patients, but show favorable response to treatment if properly controlled." In my experience if proper precautions be taken by observing any warning symptoms, such as blurring of vision, light flashes, contraction of visual fields, or disk changes, one may administer tryparsamid in moderate doses, say one gram weekly, with comparative safety. In a tabetic I observed a neuroretinitis in one eye after four injections of tryparsamid, which cleared; a month later two more injections were given and the neuritis recurred, but again cleared without nerve damage. The patient was considered unfit for tryparsamid therapy. The record is defective in this case as to the amount of the dosage, but it was probably three grams, which was the dosage generally employed when the drug was first used. In a case of neurosyphilis of taboparetic type, in which the right disk showed temporal pallor and vision was slightly, if any, affected, tryparsamid therapy was instituted after careful consideration and consultation with an oculist. The first series given consisted of four doses of one gram each. After an appreciable interval, with no untoward ocular manifestations, he has lately received seven doses of one gram each with no bad after-effects. As this patient had markedly defective vision in the

left eye since an injury in childhood, the situation was not particularly favorable for the administration of tryparsamid, but it appeared to be the drug of choice, especially so as the usual neurosyphilitic remedies had been tried with a general depressing physical and mental effect. This case favors the idea that an adverse neurotrophic action on the optic nerve is not necessarily increased by a degeneration of the nerve.

OPTIC SYNDROMES

Of optic syndromes I shall discuss but two, namely, the temporal lobe syndrome and the Foster-Kennedy syndrome.

Lesions of the temporal lobe are characterized by peculiar psychological states, hallucinations of taste and smell, fits, and in right-handed individuals when the left temporal lobe is involved, by a particular form of aphasia: amnestic aphasia for nouns, or naming aphasia, a symptom of sensory aphasia frequently mistaken for and confused with motor aphasia, leading to a false localization. By actual invasion, pressure or edema, temporal lesions may involve secondary optic neurones, producing a homonymous field defect of quadrant or complete pattern on the side opposite the lesion. Attention is particularly drawn to the frequency and importance of naming aphasia, which, when associated with field defects in right-handed individuals, is, in my experience, a most dependable and precise localizing symptom.

REPORT OF CASES

In my one hundred five cases of brain tumor occur five verified temporal brain tumors, exhibiting naming aphasia. Three examples follow:

CASE 1.—A young university instructor of languages while conducting his classes became much embarrassed by inability to recall nouns in Sanskrit; he then lost the power to recall names of common objects and the ability to name objects by sight. Visual hallucinosis for colors was present and an upper right quadrant visual defect found. A psychological state of unreality was well described by the patient. On operation a gliosarcoma was found involving a large portion of the left temporosphenoidal lobe.

* * *

CASE 2.—A case in which the great importance of a naming aphasia was not sufficiently evaluated is illustrated in the record of a young, right-handed married woman, presenting the tumor triad of papilledema, headache, nausea and vomiting. At the onset six months previously she complained of severe pain in the left eye and later of blurred vision and inability to see objects to her left unless she would turn her head to the left. Five months after onset involuntary tremors, weakness, and spontaneous past pointing developed in the right upper extremity; then followed speech difficulties: according to her husband she could not "marshal the right words to express ideas." Severe pain persisted in the left eye. Physical examination revealed a bilateral papilledema of four diopters, enlarged right pupil, a slight right-sided facial palsy of cortical type, and suspicious bilateral pyramidal tract signs in the lower extremities. Smell was doubtfully present in the left nostril, present in the right. Sight was not markedly defective, and no impairment of visual fields was found for form, although the field test was rendered doubtfully accurate by reason of speech difficulties. These consisted of a marked naming aphasia and paraphasia and defects in reading and writing, although a complicated verbal command was quite well executed and articulation was excellent. The physical findings pointed toward a left hemisphere lesion, which was sought for in the lower left fronto-Rolandic area; but the tumor, a large dural endothelioma weighing sixty-eight and a half grams, was located and removed from underneath the left temporosphenoidal lobe.

* * *

CASE 3.—A further example of the localizing value of amnestic aphasia was recently observed in the case of a right-handed child, age five, with an intracranial pressure syndrome in which the diagnosis apparently lay between encephalitis, abscess, brain tumor, or subdural hematoma. The disks showed a distinct papilledema and the cerebrospinal fluid was under pressure. With the exception of a slight ataxia and falling reaction there were no localizing symptoms. The mother related that at times there was difficulty in remembering names. In testing the child for naming aphasia, he was unable to recall the name of a pocket-knife when shown one and its use demonstrated. At times he was able to name common objects, at other times not. Ventriculograms were about to be made when the child developed a state of collapse. On puncture a brain abscess was found in the left temporal lobe.

Foster Kennedy[15] in 1911 described a syndrome due to an expanding lesion of the frontal lobes, reporting five verified cases and one clinical case. The symptom complex consisted of a "true retrobulbar neuritis with the formation of a central scotoma and primary optic atrophy on the side of the lesion, together with concomitant papilledema in the opposite eye." In three of the cases papilledema did not precede the retrobulbar neuritis on the side of the lesion.

REPORT OF A CASE

In my brain tumor series of one hundred five cases I had not observed this eye syndrome prior to the following case:

A male, age nineteen, was examined by an oculist in April 1930 for impaired vision in the left eye. A central scotoma was found in this eye; the right eye was normal. In August the right eye developed papilledema and the left disk showed pallor; vision in the left eye had diminished to slight peripheral vision. X-rays of the skull showed marked atrophy of the inner table and separation of the sutures. The sella was practically completely destroyed with possible remnants of the right anterior clinoid. Just above the left orbital plate there was some abnormal calcification. With the exception of impairment of smell in the left nostril the general neurological examination and serological tests were not noteworthy. A brain tumor was diagnosed in the left frontal lobe. This lobe and the first and second cranial nerves were explored; a marked hydrocephalus and ballooning of the cisternal system was found, but no tumor. As a result of this exploration a third ventricle tumor was considered probable. In a stormy convalescence the ventricles were repeatedly tapped, with the result that the fluid pressure finally returned to normal. Both eyes progressed to blindness with atrophy of both disks, more marked, however, on the left. It is highly improbable that a tumor could have been overlooked in the frontal lobe; moreover, the finding of a nonexpansive tumor in this locality would have been fortuitous, but not a true explanation of the syndrome.

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REFERENCES

1. Wildbrand and Saenger: J. F. Bergmann, Wiesbaden, Neurologie des Auges, Vol. iv, 1909.

2. Claude, H., Lamarche, A. and Durbar, J.: Soc. d'Ophthal. de Paris. Seance 20, October 1928. Bull. de la Soc. d'Ophthal., 40:384, 1928.

3. Dupuy-Dutemps: La Semaine des Hôp. de Paris, p. 532, 1928.

4. Tilney, F.: The Supra-Optic Canal in Its Relation to an Interpretation of Choked Disc, Neurol. Bull., 1:265 (June), 1918.

5. Farisotti: Rivista Italian di Ottalmol., Ann. VI, F. 2-9, 1910.

6. Wilson, S. A. K.: Modern Problems in Neurology, Edward Arnold Co., p. 351, 1928.

7. Ingvan, Sven: On the Pathogenesis of the Argyll Robertson Phenomenon, Bull. Johns Hopkins Hospital, 43:363, 1928.

8. Bing, Robert: Gehirn und Auge. J. F. Bergmann, Wiesbaden, 1914.

9. Spiller, W. G.: The Argyll Robertson Phenomenon, Arch. Neur. and Psych., 24:566 (Sept.), 1930.

10. Mehrtens, Henry G., and Barkan, Otto: Researches on the Pupillary Reactions in Epidemic Encephalitis, Trans. Am. Neur. Assn., p. 361, 1923.

11. Walker, Clifford B.: Retrobulbar Neuritis and Multiple Sclerosis, Cal. and West. Med., 34:5, 1931.

12. Sands, Irving J.: Amaurosis in Epidemic Encephalitis, J. A. M. A., 92:1241 (April), 1929.

13. Woods, Alan C., and Moore, J. E.: Visual Disturbances Produced by Tryparsamid, J. A. M. A., 82:2105 (June), 1924.

14. Cady, Lee D., and Alvis, B. Y.: The Use of Tryparsamid in Patients With and Without Ocular Lesions, J. A. M. A., 86:184 (Jan.), 1926.

15. Kennedy, Foster: Retrobulbar Neuritis as an Exact Diagnostic Sign of Certain Tumors and Abscesses in the Frontal Lobes, Am. Jour. Med. Sc., 142:355, 1911.

DISCUSSION

OTTO BARKAN, M. D. (490 Post Street, San Francisco).—Doctor Schaller's paper is so comprehensive that I shall touch on but a few points: Among other things I have also been struck by the relative rarity of papilledema in cases of brain abscess. In regard to the Argyll Robertson pupil, the idea is still prevalent that this sign stipulates an absent pupillary light reflex. Today the slightest diminution of the light reflex—and this can be measured numerically in relation to the norm by means of the Hess pupilloscope—in the presence of a normal accommodation-convergence reaction of the pupil constitutes a positive Argyll Robertson sign. Thus a very early diagnosis can be made and this not infrequently at a time when other signs and symptoms are absent or are not in themselves enough to establish a diagnosis. Herein lies the modern significance of the sign. Formerly a fully developed Argyll Robertson pupil was regarded as one of the early signs of neurosyphilis, but as the result of the neurological and serological advances of recent years it is no longer of such value. The definite diagnosis of the Argyll Robertson pupil in its earliest stages and its differentiation from other pupillary anomalies are, however, of the greatest value. The slightly serrated irregularity of this pupil which can usually be observed by transillumination or by the corneal microscope also serves to establish the nature of the pupillary anomaly and should always be looked for. The Argyll Robertson sign still continues to be the subject of lively discussion as it has been for over half a century. On one occasion late in the career of Argyll Robertson the sign was being discussed at length in his presence by a group of admirers and former students. When no satisfactory conclusions seemed probable, one of his former students closed the discussion with the remark that "In any case, I would rather be an Argyll Robertson pupil than have one."

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JOSEPH L. MCCOOL, M. D. (450 Sutter Street, San Francisco).—Doctor Schaller's paper opens up an almost unlimited opportunity for discussion, yet one finds it difficult to add anything to what he has said.

I am reminded, however, of a little patient with primary optic atrophy which I had the opportunity of examining about six or seven years ago. At the time I saw her she was between three and four years of age. She was a seven months' child and one of twins. The first intimation the parents had that her vision was impaired was a propensity for bumping into furniture and falling over objects. As the twin sister had no trouble in orientation the parents suspected that there must be something wrong, but did not attribute the disability to faulty vision.

As the family were intimate friends of mine, I asked them to let me examine the child's eyes, which they did. The first glimpse of the fundus confirmed my suspicions that there was a congenital impairment of the visual field, for both optic nerves were atrophic. A later examination under an anesthetic confirmed the first examination. Her twin sister, as well as an older brother and sister, all had normal eyegrounds although the oldest sister had a convergent squint with no amblyopia. The squint was corrected with glasses.

I did not have an opportunity to examine the child again as the parents never requested another examination and I was reluctant to suggest it. For that and family reasons a serological examination of the blood and spinal fluid was not made. In the absence of this, lues of the central nervous system and infantile or juvenile tabes could not be absolutely excluded, but I do not believe that syphilis was the cause of the atrophy.

I have always considered this one of those rare cases of abiosis or abiotrophy—lack of vital force affecting the optic nerves in one of immature twins.

RABIES—ITS HISTORY IN NEVADA*

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SO far as known the far West was free from rabies until 1909, at which time it made its appearance in Southern California, having been introduced directly from the East. Although recognized at that time, no effective steps were taken to eradicate it and the disease gradually spread, traveling northward through California and being introduced into Oregon in 1912 by a sheep dog taken across the mountains from Redding, California, to Wallowa County, in that state, where this infected dog, in a fight with a coyote, first introduced the disease among wild animals.

ADVENT OF RABIES IN NEVADA

Later the disease worked southeast, involving Idaho, Nevada, and Utah. Traveling by this devious route, the disease did not reach Nevada until April, 1915, although during its passage northward through California it appeared at times very close to the California-Nevada line, but did not cross into and establish itself in the latter State, presumably because at that time the disease was confined to dogs, not involving wild animals, until Oregon was reached as above.

Nevada appears to have finally been invaded by three routes, the first authenticated appearance of rabies being at the northern border of Humboldt County in April, 1915, the disease having crossed the state line at that point from Oregon. Later invasions took place from northeastern California into Washoe County and from Idaho into Elko County. Once introduced into the northern part of the state, the disease was spread by means of

* From the Nevada State Veterinary Control Service.

* Read before the Washoe County Medical Society, March 8, 1932.